DEPARTMENT OF MEDICINE

EFFECT OF HYPERTENSION AND ASSOCIATED RISK FACTOR ON KIDNEY SIZE IN MIDDLE AGED ADULT

THESIS
FOR
DOCTOR OF MEDICINE
(INTERNAL MEDICINE)

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RAVINDRA MOHAN KATIYAR

Dedicated to
Respected Teachers, parents,
Friends & my supportive
family members

DEPARTMENT OF MEDICINE

M.L.B. Medical College, Jhansi (U.P.)

CERTIFICATE

This is to certify that the work entitled "**EFFECT OF HYPERTENSION AND ASSOCIATED RISK FACTOR ON KIDNEY SIZE IN MIDDLE AGED ADULT**" which is being submitted as thesis for M.D. (Medicine) Examination 2006, Bundelkhand University, Jhansi, has been carried out by **Dr. Ravindra Mohan Katiyar** under my direct supervision and guidance. The techniques consumed in the preparation of this thesis were undertaken by the candidate himself and the observations recorded were checked and verified by me from time to time.

Dated : 28/10/05

Dr. P.K. Jain
M.D., MNAMS
Professor & Head,
Department of Medicine,
M.L.B. Medical College,
Jhansi (U.P.)
(Guide)

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Dated : 28/10/05

Dr. N.S. Sengar
M.D.,D.M.(Nephrology)
Associate Professor,
Department of Medicine,
M.L.B. Medical College,
Jhansi (U.P.)
(Co-Guide)

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Ravindra Mohan

Date : 28/10/05

Ravindra Mohan Katiyar

CONTENTS

Introduction

INTRODUCTION

Hypertension is amongst the most common imported health problem throughout the world. It is a major cause of mortality and morbidity in society but it exact prevalence is not known because of large number of representing submerged portion of iceberg.

Hypertension is one such disease which is slow, secret and silent threat to people, thoughout the world with the potential to cause serious and organ damage. Hypertension affect heart, kidney eyes, brain and almost all organ of the body.

Hypertension is the disease that is usually diagnosed when it has already cause major damage to the body. system or persist as acute complication which may lead to death.

Hypertension is readily detectable, easily treatable and leads to lethal complications it left untreated.

In the VIIth of the Joint National committee on Prevention, detection, evaluation and treatment of high Blood Pressure (JNC VII) hypertension is classified as

BP Classification	Systolic BP, mmHg	Diastolic BP, mmHg
Normal	< 120 and	< 80
Prehypertension	120 - 139 or	80 - 89
Stage 1 Hypertension	140 - 159 or	90 – 99
Stage 2 Hypertension	≥ 160 or	≥ 100

Although many condition and disorder are known now that causes increased blood pressure, the cause remain unknown in over 90% of cases. So these patients with no definite cause are said to have primary, idiopathic or essential hypertension. Several mechanism have been described in patients with essential hypertension, like dysfunction of sympathetic nervous system, renin-angiotensin system defect, increase salt sensitivity, sodium transport defect and certain risk factor like obesity, smoking, alcoholism and dyslipidemia.

Other than this major group of patients these are patient in whom there is an obvious cause leading to hypertension. These are cases of secondary hypertension cause of which are :-

1. Renal
    - Acute / chronic glomerulonephritis

- Pyelonephritis
- Renal artery stenosis
- Renal tumour and cysts

2. Endocrinal
   - Cushing's syndrome
   - Primary aldosteronism
   - Pheochromocytoma
   - Oral contraceptive
3. Others
   - Coarctation of aorta
   - Toxaemia of pregnancy

Untreated hypertension increases the risk of vascular damage involving both small (resistance) arteries and arterioles and large (canduit) arteries. These leasions lead to cardiac, renal and cerebrovascular morbidity and mortality. The incidence of these different lesions is also dependent upon the level of other risk factor such as plasma cholesterol, diabetes, alcoholism, smoking and obesity.

Kidneys are important target of hypertension induced organ damage. Urine analysis, creatinine clearance, ultrasonic kidney size,

pyelogram and angiogram are relatively normal in patients with essential hypertension.

Abnormalities of standard kidney function test in patients with long standing, poorly controlled hypertension in the absence of intercurrent primary diseases of the kidney are attributable to benign nephrosclerosis. The development of renal damage in hypertension is commonly hearded by proteinuria. Under these circumstances low grade proteinuria (<1gm/day) and creatinine clearance may fall and kidney may shrink. Advance nephrosclerosis is characterized by symmetric reduction in kidney size and increase echogenecity on renal ultrasonography.

Malignant hypertension of lead to renal insufficiency within a few years. Mostly as a consequence of fibrinoid necrosis of small renal arteries.

Ultrasonography is an accurate method of estimating kidney size.

To assess abnormalities in renal size, maximum bipolar length measurement is most practical and recommended method.

* Normal adult kidney size is 9-12 cm in length.

* Healthy men have larger kidney than healthy women.

- In healthy adult right kidney is slightly smaller than left.
- Kidney length diminished by approximately 0.5cm per decade.
- Renal length correlates best with body height.

Review of Literature

REVIEW OF LITERATURE

It was in 1628 when Sir William Harvey demonstrated the blood circulation and showed that the heart is central organ to pump the blood to different organs.

In 1973 Clegyman reverent Stephen Hales described the result of his experience on eth blood pressure of a man and he measured intra arterial pressure for the first time. He expressed pressure in terms of weight of blood itself, a time when there was no way to measure blood pressure directly. In man blood pressure was estimated to be about 7.5 feet of blood which corresponds to about 176 mmHg.

1847 Harrison devised the first sphygmomanometer.

J Faivere in 1856 measured blood pressure accurately.

The first book devoted to blood pressure was written by Walenberg in Berlin in 1880.

Korotokoff in 1905 introduced auscultatory technique for blood pressure measurement and suggest that sound, heard over the arteries, distal to cuff should be used to indicate blood pressure.

Frank (1911) first recognized the so called primary or essential hypetension.

Theodore C. Janeway (1913) stated abnormal pressure above 160 mmHg after study of 7872 cases.

Globlatt et al (1934) were the first to produce experimental hypertension by partially constricting the renal artery in a dog.

Bordely et al (1951) concluded that the point of complete cessation of sound is best index of diastolic blood pressure.

World health Organization (1962) recommended that both muffing and cessation of sound should be recoded for diastolic blood pressure.

The definition of hypertension is based primarily on the blood pressure levels that have been established to define those who have an increase risk of developing a morbid cardiovascular event and / or will clearly benefit from medical therapy. These definition should consider not only the levels of diastolic pressure but also systolic pressure age, sex and race.

The current classification based on systolic and diastolic blood pressure levels – High normal diastolic blood pressure (85-89 mmHg) is now considered to be category separate from normal. Persons with blood pressure in this range have a higher risk of developing hypertension and cardiovascular complications and therefore should be

considered for nonpharmacologic approaches to prevent hypertension and its complications.

Hypertension is very common in persons over 65 years of age. Approximately two thirds of the aging population have a systolic pressure of 140 mmHg or higher or a diastolic pressure of 90 mmHg or more or both. In most older patients the systolic pressure alone is elevated, a condition known as isolated systolic hypertension. This type of hypertension increase the risk of cardiovascular complications such as congestive heart failure stroke ischaemic heart disease and left ventricular hypertrophy (Working group on Hypertension in Elderly 1986).

CLASSIFICATION OF HYPERTENSION

Hypertension is defined as SBP of 140 mmHg or greater DBP of 90 mmHg or greater or taking antihypertensive medication. The objective of identifying and treating high blood pressure is to reduce the risk of cardiovascular disease and associated morbidity and mortality.

"The Seventh Report of the Joint National Committee on Prevention. Detection. Evaluation, and Treatment of High Blood Pressure" provides a new guideline for hypertension prevention and management. The following are the key messages: (1) In persons older

than 50 years systolic blood pressure (BP) of more than 140 mm Hg is a much more important cardiovascular disease (CVD) risk factor than diastolic BP; (2) The risk of CVD beginning at 115/75 mm Hg doubles with each increment of 20/10 mm Hg; individuals who are normotensive at 55 years of age have a 90% lifetime risk for developing hypertension; (3) Individuals with a systolic BP of 120 to 139 mm Hg or a diastolic BP of 80 to 89 mm Hg should be considered as prehypertensive and require health-promoting lifestyle modifications to prevent CVD; (4) Thiazide-type diuretics should be used in drug treatment for most patients with uncomplicated hypertension either alone or combined with drugs from other classes. (5) Most patients with hypertension will require 2 or more antihypertensive medications to achieve goal BP (<140/90 mm Hg or <130/80 mm Hg for patients with diabetes or chronic kidney disease); (6) If BP is more than 20/10 mm Hg above goal BP consideration should be given to initiating therapy with 2 agents, 1 of which usually should be a thiazide-type diuretic; and (7) The most effective therapy prescribed by the most careful clinician will control hypertension only if patients are motivated.

JNC VII provides a classification of blood pressure for adults (age 18 or older). These criteria are for individuals who are not taking antihypertensive medication and who have no acute illness. This

classification is based on the average of two or more blood pressure reading taken in accordance with the following recommendations at each of two or more visits after an initial screening visit. When SBP and DBP fall into different categories the higher category should be selected to classify the individuals blood pressure. The classification is slightly modified from JNC VI report, a new category designated prehypertension has been added, and stage 2 and 3 hypertension have been combined in JNC VII.

Category	Systolic (mmHg)	Diastolic (mmHg)
Normal	< 120	< 80
Prehypertension	120 - 139	80 – 89
Hypertension Stage I Stage II	 140 – 159 ≥ 160	 90 – 99 ≥ 100

Systolic hypertension is determined of arterial pressure influenced on cardiovascular morbidity.

- Isolated Systolic Hypertension : is defined as systolic blood pressure of 140 mmHg or more and a diastolic blood pressure of < 90 mmHg. Patients with isolated systolic hypertension have a 2.5

folds increase in their cariovascular mortality rates when compared with individuals with similar diastolic pressure but normal systolic pressure (Williams GH 1991).

- Isolated Diastolic Hypertension or Decapitate Hypertension : is extremely rare and is seen only with mild elevation of diastolic pressure (e.g. 120/100 mmHg). It is usually found in children and young adults.

- Malignant Hypertension : Sudden rise of very high pressure above 200 mmHg with Papilloedema usually accompanied by retinal haemorrhages and exudates, pathologically characterized by necrotizing arteries and fibrinoid degeneration. It is papilloedema not the absolute pressure level that defines this condition.

- Accelerated Hypertension : Signifies a recent increase over previous hypertensive levels associated with evidence of vascular damage on fundoscopic examination but without papilloedema.

- Labile Hypertension : These patients are said to have labile hypertension who sometimes, but not always have arterial pressure within hypertensive range.

CLASSIFICATION ACCORDING TO EXTENT OF ORGAN DAMAGE

According to National High Blood Pressure education programme working group WHO 1978.

STAGE I

- No Objective signs or organic change in the cardiovascular system.

STAGE II

- Left ventricular hypertrophy
- Retinal involvement
- Proteinuria

STAGE III

- Evidence of signs and symptoms of organ damage due to hypertension.
- Heart left ventricular hypertrophy, left ventricular failure.
- Brain- cardiovascular haemorrhage, hypertensive encephalopathy.
- Eye- retinal haemorrhage and exudates with or without papillodema.

CLASSIFICATION ACCORDING TO ETIOLOGY

A. Primary or essential or Idiopathic hypertension

B. Secondary hypertension

A. ESSENTIAL OR PRIMARY HYPERTENSION

The cause of elevated arterial pressure remains unknown in most of the cases. In general population about 90-95% hypertensive constitute this group. Patients with arterial hypertension with no definable cause are said to have primary, essential or idiopathic hypertension. Development as hypertension depends on the interaction between genetic predisposition and environmental factors. How his interaction occurs is still incompletely underst6ood. It is known that hypertension is accompanied by functional alterations of sympathetic nervous system, the kidney, the rennin – angiotensin system and other humoral mechanisms.

SYMPATHETIC NERVOUS SYSTEM : Sympathetic nervous system may play a major role in initiating essential hypertension and may contribute to hypertension related to hyperdynamic circulatory states.

Several authors have reported increased concentrations of norepinephrine in the plasma of patients with essential hypertension (Esler M et al, 1989). Recent confirmation comes from studies on sympathetic activity recorded directly from sympathetic nerves of superficial muscle in patients with essential hypertension (Anderson EA et al, 1989; Mancia G et al, 1993).

RENAL MECHANISM : Renal mechanism have often been implicated in the pathogenesis of hypertension, either through an altered pressure natriuresis leading to sodium retention or through an altered release as pressure factors (such as rennin) (Cowley AW, Roman R, et al, 1986).

RENNIN – ANGIOTENSIN SYSTEM : The rennin angiotensin system has a major role in the physiological control of blood pressure and sodium balance. The range of plasma rennin activities observed in hypertensive subjects is broader than in normotensive individuals.

*** Low renin essential hypertension** – Approximately 20% of patients who by all other criteria have essential hypertension have suppressed rennin activity. Though these patients are not hypokalemic they have been reported to have expanded extracellular fluid volumes, and it has been suggested but not proved that they have sodium retention and

rennin suppression due to excessive production of an unidentified mineralocorticoid. (Harrison's principle's or Internal Medicine 16th edition).

* **Non modulating essential hypertension** – Another subset of hypertensive patients have an adrenal defect opposite to that observed in low- rennin patients – a reduced adrenal response to sodium restriction. In these individuals, sodium intake does not modulate either adrenal or renal vascular response to angiotensin II. Hypertensive in this subset have been termed normodulators because of the absence of the sodium mediated modulation of target tissue response to angiotensin II. These individuals make up 25-30% of hypertensive population, have plasma renin activity levels that are normal to high, and have hypertension that is salt sensitive because of a defect in the kidney's ability to excrete sodium appropriately.

* **High – rennin essential hypertension** – Approximately 15% of patients with essential hypertension have plasma rennin activity levels above the normal range elevated rennin levels and blood pressure in these patients may be secondary to an increase in adrenergic system activity (Harrison's Principle's of Internal Medicine 16th edition).

STRUCTURAL CARDIOVASCULAR ADAPTATION

The increased load on the vascular system caused by high blood pressure and activation of growth factors leads to structural adaptations with narrowing of the arteriolar lumen and an increase in the media wall ratio. This amplifies resistance to blood flow and increase vascular responsiveness to vasoconstrictor stimuli (Folkow B et al, 1992).

Cardiac structural adaptations consist of thickening of the left ventricular wall in response to an increase after load (concentric hypertrophy) and an increase in left ventricular diameter (Koren MJ et al, 1991). Both vascular and cardiac structural adaptations act as amplifiers of the hemodynamic pattern of hypertension and as initiators of several of the complications of hypertension (Korner PL et al, 1994).

ENDOTHELIAL DYSFUNCTION

New studies have shown endothelium involvement in the conversion of angiotensin I to angiotensin II, in kinin inactivation and in the production of endothelium derived relaxing factor or nitric oxide. Endothelium plays a role in the local hormonal and neurogenic control of vascular tone and the haemostatic process. Endothelium also release vasoconstrictive agents, including endothelin that may be implicated in some of vascular complications of hypertension (Lucher TF et al, 1990).

B. SECONDARY HYPERTENSION

This group constitutes 5-10% patients of hypertension

1. Renal

A. Renal parenchymal disease

- Acute glomerulonephritis
- Chronic nephritis
- Polycystic disease
- Diabetic nephropathy
- hydronephrosis

B. Renovascular

- Renal artery stenosis
- Intrarenal vasculitis

C. Renin producing tumors

D. Primary sodium retention (Liddle's syndrome, Gordon's syndrome)

2. Endocrine

A. Acromegaly

B. Hypothyroidism

C. Hyperthyroidism

D. Hypercalcemia (hyperparathyroidism)

E. Adrenal

I. Cortical

- Cushing's syndrome
- Primary aldosteronism

- Congenital adrenal hyperplasia

II. Medullary

- Phenochromocytoma

F. Extraadrenal chromaffin tumors

G. Carcinoid

3. Coarctation of aorta

4. Pregnancy induced hypertension

5. Neurological disorders

A. Increased intracranial pressure

- Brain tumor
- Encephalitis
- Respiratory acidosis

B. Sleep apnea

C. Acute porphyria

D. Guillain – Bare syndrome

E. Familial dysautonomia

6. Acute stress

- Post operative
- Sickle cell crises
- Psychogenic hyperventilation

7. Increased intravascular volume

8. Diet and drugs

- Alcohol

- Estrogens
- Glucocorticoids
- Sympathomimetics
- Amphetamines
- MAO inhibitors
- Licorice
- Cocaine

9. Isolated Systolic Hypertension

A. Increased cardiac output

- Aortic valvular insufficiency
- A-V fistula
- Patent ductus
- Thyrotoxicosis
- Paget's disease of bone
- Beri-Beri

B. Aortic arteriosclerosis

FACTORS DETERMINING BLOOD PRESSURE

Increasing attention is being paid to an examination of factors that coorelated with blood pressure levels during childhood in the hope of identifying which of these are determinants of the risk in blood pressure in particular, the frequency observed, but until recently poorly studies, changes in blood pressure with the onset of puberty in now being investigated intensively (Level and Harrap, 1991). The importance of

recognition of such factors lies in the generally accepted view that, if they were identified in childhood and adolescence, primary prevention, of adult onset hypertension might become a realistic public health and clinical goal.

Epidemiologic factors related to blood pressure levels in children and adolescents.

GENETIC

- Parental and sibling blood pressure level.
- Erythrocytes sodium flux.
- Haptoglobin phenotype 1-1
- Increased salt sensitivity in blacks

ENVIRONMENTAL

- Socio –economic status
- Rural versus urban residence
- Pulse rate
- Small gestational age
- Exercise

MIXED GENETIC AND ENVIRONMENTAL

- Height

- Weight
- Body mass
- Obesity and response to sodium
- Sodium and Potassium excretion
- Stress
- Skinfold thickness

Smoking

- Ambulatory blood pressure monitoring recognized the major pressure effect of smoking (Mann et al, Decaris et al, Goppeli et al, 2000).
- Smokeless tobacco and cigars, if their smoke is inhaled also may raise blood pressure.
- The noxious cardiovascular effects of smoking also involve a worsening of lipid status an increase in central obesity, which in turn may be involved in worsening of insulin resistance. Thus, hypertensive who use tobacco must be repeatedly and umambigously told to quit and given assistance in doing so.

ALCOHOL

Moderate alcohol consumption, less than 1 Oz. Of ethanol per day does not increase the prevalence of hypertension. Heavier drinking clearly exerts a pressor effect that make alcohol abuse the most common cause of reversible hypertension (Alderman MH, 1994).

Excessive alcohol intake is an important risk for high blood pressure (Stamler J, 1997) can cause resistance of antihypertensive therapy (Puddey IB, 1992).

Those who drink beverages containing alcohol consumption should be elicited from patients. Those who drink beverage containing alcohol should be counseled to limit the daily intake to no more than 1 Oz (30ml) of ethanol – For eg. Ounces (720 ml) of Beer 10 ounces (300 ml) of wine and 2 ounce (60 ml) 100 proof whisky.

Although acute alcohol intake causes peripheral vasodilation with a consequent fall of blood pressure (Altura and Altura 1982), chronic consumption increases the blood pressure in a dose dependent manner (Keil et al, 1993).

The pressure effect of alcohol seems to be enhanced by obesity, advanced age and by high stress occupation (Vandongen and Pussey

1994). The amount of alcohol required for pressor effects to occur is not exactly known and underlies certainly great individuals variability.

Obesity

Hypertension is more common among obese individuals and probably adds to their increased risk of developing ischemic heart disease. Adiposity as measured by subcapular skinfold thickness is major controllable contributor to hypertension (Sonne – Holm S., Sorensen TIA & Schnohr P, 1999).

Baseline systolic blood pressure, current weight and weight gain are significantly associated with current systolic blood pressure and hypertension. The initial systolic blood pressure, pressure at adolescence, current weight and gain in weight are important determinantes of risk of high blood pressure in study performed by Young et al, 1993.

Increased age more than 50 yrs, high body mass index B.M.I. (More than 23) and hyperglycemia show significant association with high systolic and diastolic blood pressure (Sayeed M.A. et aI, 1994).

Children seem particularly vulnerable to hypertensive effects of weight gain (Liberman, E. 1994). Therefore avoidance of childhood

obesity with the hope of avoiding subsequent hypertension seems important.

The deposition of excess fat in the upper part of body (Visceral/ Abdominal) as evidence by a waist circumference of 34 inches (85 cm) or greater in women or 39 inches (98 cm) or greater in men, also has been associated with the risk for hypertension, dyslipidemia, diabetes and coronary heart disease mortality (Pouliot MC et a11994).

DYSLIPIDEMIA

It has been proposed that dyslipidemic hypertension is part of a, distinct metabolic syndrome related to insulin resistance. Dyslipidemia and hypertension are usually associated with obesity and diabetes mellitus.

High blood pressure has been associated with elevated atherogenic lipid fractions. There are biological interrelation between blood pressure and blood lipids they may influence the mechanisms where by blood pressure is associated with risk of coronary heart disease. Total and non HDL - cholesterol levels increases significantly with increasing systolic or diastolic blood pressure in both sex.

Naa, Thelle D.S. have shown that in men this association between blood pressure and total cholesterol level decreases with age, where as

in women, it increase with age. Body mass index modified the relation, where as smoking physical activity and alcohol consumption and little influence on this association.

In Halland Green and associates indicated that the average cholesterol level among healthy individual has increased considerably during the lost fourty years. Kinsell and associates have confirmed Green's results.

Pauletto et al have reported that catechal amines increase polylipoidization of aortic smooth muscle cells, increased cholesterol of arterial wall, also induces free fatty acid transformation into triglycerides and increase in very low density lipoprotein and decrease in high density lipoproteins levels.

Dyslipidemia is a major independent risk factor for coronary artery disease therefore dietary therapy and if increasing, drug, therapy for dyslipidemia are an important adjuvant to antihypertensive treatment. In randomized controlled studies, diets varying in total fat have had little if any effect on blood pressure. Large amounts of Omega 3 fatty acids may lower blood pressure (Toft I, Benaa KH et al, 1995).

NCEP (ATP III) Guidelines for lipids abnormalities –

TOTAL CHOLESTEROL :

< 200 mg/dl	Desirable
200 – 239 mg/dl	Borderline high
> 240 mg/dl	High

LDL :

< 100 mg/dl	Optional
100 – 129 mg/dl	near optimal / above optimal
130 – 159 mg/dl	Border Line high
160 – 189 mg/dl	High
> 190 mg/dl	Very high

HDL :

< 40 mg/dl	Positive risk factor for CAD
≥ 60 mg/dl	negative risk factor for CAD

TRIGLYCERIDES :

< 150 mg/dl	Normal
150 – 199 mg/dl	Borderline high
200 – 499 mg/dl	High

DIABETES

Hypertension and diabetes coexist more commonly than predicted by chance. They feed on each other to markedly accelerate cardiovascular damage.

51% of insulin – dependent diabetics and 80% of the non insulin dependent diabetics had blood pressure above 140/90 mmHg (Tarnow L, Rossuing P, et al, 1994).

Not only hypertension more common in diabetes, but it also tends to be more persistent, with less of the usual nocturnal fall in pressure (Lurbe A, Pascual J. M. et al 1993). The absence of a nocturnal fall in blood pressure may reflect aiutonomic neuoropathy or incipient diabetic nephropathy (Serrut. G. Bouhanik B, et al, 1998).

The presence of hypertension increases all the microvascular and macrovascular complications seen in diabetes. Even at the initial presentation of diabetes, the presence of hypertension is associated with about a doubling of the prevalence of microalbuminuria, left ventricular hypertrophy and electrocardiographic signs of myocardial ischemia (J. hypertens 11 : 309,1998).

ELECTROLYSIS

Sodium in the form of sodium chloride or tablet salt, is linked to levels of old pressure. Individual response of blood pressure to variation in sodium intake differs widely as groups, African, Americans older people and patients with hypertension or diabetes are more sensitive to changes in dietary sodium chloride than one others in the general population (Weinberger MH, 1996).

Midgley JP et al (1996): An analysis of 17 published randomized controlled trials involving patients age 45 or older with hypertension found

an average decrease of 6.3/2.2 mmHg with a urinary sodium reduction of 95 m mol/day.

Mac Greger GA et al : A double blind study of three sodium intake. In small but well controlled study: the fall in blood pressure was shown to be 8/5 mmHg on a daily sodium of 100mmol and 16/9 mmHg on a 50 m mol/day intake.

Feldman RD showed even if the blood pressure does not fall with moderate degrees of sodium restriction, the patients may still benefit improved beta adrenergic responsiveness.

Whelton PK (1997) showed that high dietary potassium intake may protect against developing hypertension and improve blood pressure control in patients with hypertension.

CAFFEINE

Caffeine may raise blood pressure acutely. Tolerance to the pressure effect develops rapidly and no direct relationship between caffeine intake and elevated blood pressure has been found in most epidemiologic surveys (Stamler) Cagginla A. W. et al, 1997).

PHYSICAL ACTIVITY AND EMOTIONAL STRESS

Sedentary life style is more responsible for increase in prevalence of hypertension in women as compared to men (Ainsworth et ai, 1998).

During physical exercise (Aerobic) the systolic pressure rise considerable and vascular compliance increases during dynamic exercise (Cameron, 994) and resting blood pressure usually falls (Dubbert, 1994).

Regular aerobic physical activity adequate to achieve at least a moderate levels of physical fitness can enhance weight loss and functional health status and reduce the risk for cardiovascular disease and all cause mortality (Pafferbouger RS,1993), (Kohhinos PH,1995).

Blood pressure can be lowered with moderately intense physical activity (40-60% of maximum 2 consumption such as 30-45 minutes of brisk walking most days of weak. When compared their more active and fit peers, sedentary individuals with blood pressure have a 20-50 percent increased risk developing hypertension (Blair SN (1984).

Emotional stress can raise blood pressure acutely. The role of stress management techniques in treating patients with elevated blood pressure is uncertain. Relaxation treating therapies and biofeedback have studied in multiple controlled trails with little effect beyond that seen in control groups (Van Mart Frans et al, 1990).

When patients even those as difficult to control as outpatients are hospitalized, their blood pressure almost always comes down mainly

because the sympathetic nervous system becomes less active (Hossmann et al, 1981).

The blood pressure usually falls considerably during sleep however, there is no evidence that sedatives or transquilizers lower (US Public Health (1965). MAO inhibitor will lower the blood pressure but their use is limited by the potential for bad pressore reactions with tyramine containing foods.

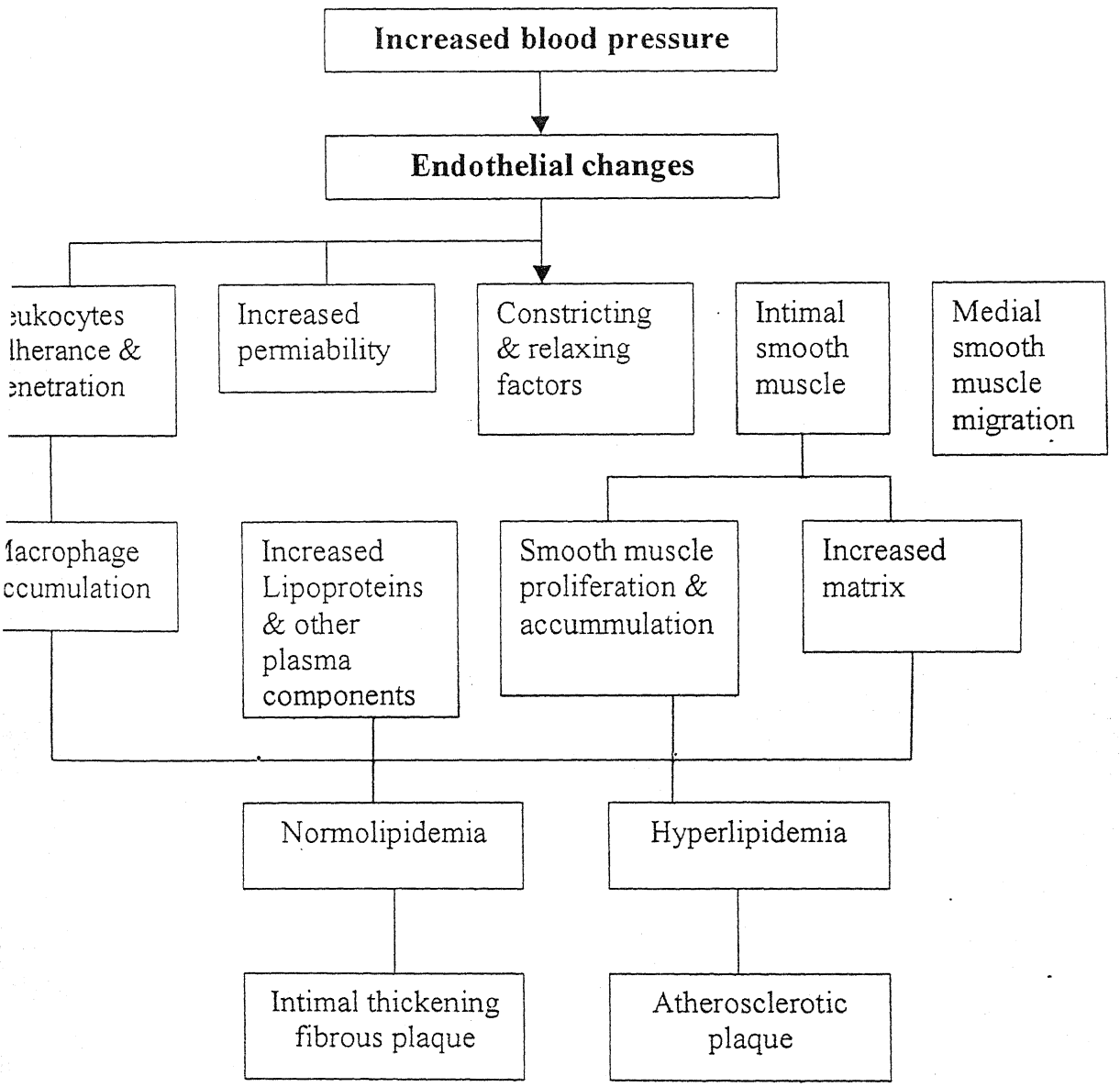
COMPLICATIONS OF HYPERTENSION

The end of the natural history of hypertension is an increased likelihood of premature disability or death from cardiovascular disease.

Before considering the specific types of organ damage. It is important to know the underlying basis for the arterial pathology caused by hypertension which as we known in turn is responsible for the target organ damage.

The pathogenesis of hypertension involves structural changes in the resistance arterioles subsurred under the terms, remodelling and 'hypertrophy'. These same changes almost certainly are also involved intimately in the development of small vessel arteriosclerosis that is responsible for the same time, high pressure accelerates large vessel atherosclerosis. Such arterial and arteriolar sclerosis may be considered the

secondary of typical combined systolic and diastolic hypertension. Whereas it is the mechanism primarily responsible for the predominantly systolic hypertension. Artherosclerotic plaques appear most commonly where the pressure is highest, such as in abdominal aorta rather than in the low - pressure pulmonary arteries.



SPECIFIC ORGAN INVOLVEMENT

In General, organ damage due to hypertenssion can be considered either 'hypertensive' or 'artherosclerotic', hypertensive complications are caused more directly by the increased level of blood pressure per se whereas the atherosclerotic complication have multiple cause, hypertension playing a variable role (Birkenhager & de Leeuw, 1988).

COMPLICATIONS OF HYPERTENSION

HYPERTENSION PER SE

- Accelerated malignant hypertension (grade III & IV retinopathy).
- Encephalopathy
- Left ventricular hypertrophy
- Congestive heart failure
- Renal insufficiency
- Aortic dissection
- Retinopathy

ATHEROSCLEROTIC

- Cerebral thrombosis
- Myocardial infraction
- Claudication syndromes

- Coronary artery disease

EFFECTS ON HEART / HYPERTENSIVE HEART DISEASE

Hypertension both accelerates the development of coronary artery disease and puts increased tension on the myocardium causing it go to hypertrophy. These conditions in turn may result in myocardial ischemia and thus ischemia copules with LV hypertrophy may lead to congestive heart failure arrythmias and sudden death (Massie et al, 1989).

LARGE VESSELS DISEASE

Hypertension is a risk factor for the development of peripheral vascular disease that usually is manifested as intermitted claudication.

Others are:

- Abdominal aortic aneurysm
- Aortic dissection- 80% cases are associated with hypertension (Spittel et al, 1993).
- Takayasu's disease - Reported most frequently in Japan and India (Ishikava, 1988).

  Large vessel disease is accompanied by a high risk of death from cardiovascular caused (Criquie et al, 1992).

CEREBROVASCULAR DISEASE

Cerebrovascular disease is the third most common cause of death after heart disease and cancer. In industrialized countries, strokes are responsible for 10-12% of all death (Bonita, 1992). About 70% of strokes are ischemic, 10% to 15% are caused by intraparenchymal haemorrhage, 5% are cause by sub - arachnoid haemorrhage and 5-15% are of unknown cause (Anderson et al, 1993). Majority of these case alongwith transient ischemic attacks (TIA) are attributed to hypertensive changes of peripheral vessels.

The risk is even greater in hypertension with other risk factor, including diabetes, smoking, atrial fibriliation,LVH (Wolfet al, 1991) blood hypervicosity (Can II et al, 1991) and a high hematocrit (Pery et al, 1992).

RETINAL CHANGES

Increasing severity of hypertension is associated with focal spasm and progressive general narrowing of the arterioles as well as the appearance of haemorrhage, exudates and papilloedema. These retinal changes often produce scotomata, blurred vision and even blindness.

EFFECT OF KIDNEY

Persistent exposure of the renal circulation to elevated intraluminal pressure result in development of intrinsic lession of the renal arterioles (Hyaline arteriosclerosis) that eventually lead to loss of function

(Nephrosclerosis). The urine analysis, creatinine clerance, ultrasonic kidney size, pyelogram and angiogram are relatively normal in patients with essential hypertension.

If the urinary sediment, blood urea nitrogen (BUN) and creatinine are normal and protein urea does not exceed 1 gm / day it can usually be assumed that the hypertension is not secondary to primary renal parenchymal disease.

Both glomerular hyperfiltration and microalbuminuria are early markers of hypertensive nephropathy (Schmeider RE, Numez 8, et al, 1990).

NEPHROSCLEROSIS IS DIVIDED INTO TWO DISTINCT ENTITIES

1. ***Beingn Arteriolar Nephrosclerosis:*** Seen in patients who are hypertensive for an extended period of time but whose hypertension has not progressed to a malignant from. Under these circumstances low grade proteinuria (<1 gm/day) and granular casts may appear, creatinine clearance may fall. Kidney size is normal to reduced with loss of cortical mass leading to fine granularity. Advanced nephrosclerosis is characterized by a symmetric reduction in kidney size and increased echogenicity on renal ultasonography. Characteristic

pathology is in afferent arterioles which have thickned walls (hyaline arteriosclerosis).

2. **Malignant Arteriolar Nephrosclerosis:** Renal failure from malignant hypertension is usually seen in a clinical context of multiple target organ decompensation (Retinopathy, encephalopathy and congestive heart failure).

Histologically two distinct vascular lesions can be seen - the first is fibrinoid necrosis and second is concentric hyperplastic proliferation of the cellular elements of the vascular wall.

Renal abnormallities include rapid rise in serum creatinine, hematuria, protenuria and red and white blood cell casts, nephrotic syndrome may be present. (Harrison's Principle of internal medicine 14th edition).

RENAL ULTRASOUND

The potential use of ultrasound as a diagnostic aid in medicine was demonstrated 1st by Hairy and Bliss in 1952.

The first report on application of diagnostic ultrasound to the kidney was given in 1954 by Holmes et al who demonstrated a kidney cyst with the aid of this new diagnostic principle.

Schiegel et al (1961) and Heap (1968) in this study of a patients with nephrolithoiasis, found 'A' - scan ultrasound examination very useful and

simple in all instances in accurately locating the kidney stone during surgery and thus simplifying its removal.

Barlyne (1961) used ultrasound to identify the lower renal pole for needle biopsy.

Holmes (1966) was the first to use diagnostic ultrasound for urinary tract by diagnosing a renal cyst. He also described general applications of ultrasound and stated that 'B' scan ultrasound is characteristic in polycystic disease.

Renal ultrasonography most readily demonstrate solitary renal cyst, polycystic kidney, renal tumours, hydronephrosis, perinephric abscess- Mount Ford et al (1997), Barnett and Morie (1992) and renal size in different clinical situation.

Page JE et al (1984) concluded in renal parenchymal disease, analysis of sonographic and histological finding showed statistically significant positive correlation between renal size and the extent of glomerular hypercellularity and crescent formation.

Avram MM, Hurtado H. (1989) in study' Renal size and function renal size was measured by ultrasonography.

RENAL SIZE

- Normal adult kidney length is 9-12 cm and width is 4-6cm(it varies

with a range of scan. renal sinus (Medullary part) is 1/3 of kidney.

- Ultrasound is generally accepted as an accurate method of measuring renal size (Absey et ai, 1997, Emamian et al 1993).
- Ultrasound may be even more accurate than measurement bases on plain radiographs, excretory urograms or renal angiograms (Ninan et al, 1990).
- For assessment of abnormalities in renal size, measurement of renal bipolar length is recommended (Emamian et al, 1993).
- Children usually have no difference in kidney size between the sexes (Dinkel et al 1983) while in adult population men have larger kidneys (Emamians et al ,1993).
- Emamian et al (1993) found the right kidney to be slightly smaller than the left in adults.
- Renal length correlates best with body height (Emmian et al 1993).
- Renal size decrease with age, (Emamian et al 1993) after middle age, kidney length diminished by approximately 0.5 cm per decade (Mc Lachlan, Wasserman P .1981).

ASSOCIATED .DIABETES MELLITUS ALSO EFFECTS KIDNEY SIZE :-

Christalansen JS et al (1981) concluded in study thatbin early phase of diabetic nephropathy kidney size increased up to twenty percent.

Dumler F, et al (1987) found in study' Renal involvement in type 2 diabetes mellitus, the renal volume was increased.

Hirsch Berg Rand Kopple JD (1989) concluded that growth hormone and insulin like growth factor are responsible for renomegaly.

Yamada H et al (1992J found out renal size reduction accompanied by the decrease of renal function in chronic renal disease and diabetic nephropathy.

Several studies have shown that in non - insulin dependent diabetes (NIDDM) the glomerular rate is elevated compared to matched non- diabetic subjects (Palmisano and Lebovitz 1989, Marre et a11992, Nowack et a11992, Vora et aI1992).

GIomeular filtration rate is increased in newly diagnosed NIDDM and significantly related to the like wise increased kidney size (Wirta and Pasternack 1995). Such a relationship has been perviously shown to occur in insulin- dependent diabetes.

Although there is a little evidence that alcohol abuse directly damage

the kidney, a wide range of renal and electrolyte/ acid base disorders are indirectly induced (De Marchi et al , 1993, Heidland et al 1985, Konchel 1981).

In most investigations acute alcohol administration did not alter glomerular filtration rate (GFR) and renal plasma flow (Kalbfleisch et al 1963). After chronic alcohol ingestion an impairment of GFR associated with renal hypertrophy in rats was found. Histopathological examination revealed interstitial edema and tubular dilatation with flattening of the epithelial lining cells (Van Theiel et al 1999).

Smoking also affects kidney size. In one study of hypertensive patients large kidney size was found in smokers (Plaivansolo, MJ, Merikanto J et al 1998). Renal size increased with the pack years smoked.

Aims & Objectives

AIMS AND OBJECTIVES

1) Whether the renal size of hypertensive subjects differs from that of control subjects.

2) Whether the difference is due to hypertension itself or risk factors associated with hypertension

Material & Methods

MATERIAL AND METHODS

The present study was hospital based study. Patients for the present study were selected to predecided selection criteria from those attending out patient department, Nephrology clinic, Cardiologic clinic, Hypertension clinic and in Medicine Wards of MLB Medical College, Jhansi. The protocol of study was explained to all patient and their verbal consent was obtained for the inclusion in the study.

SELECTION OF CONTROLS

15 normotensive person both male and female ages 41-60 years and free from renal disease and diabetes.

SELECTION OF CASES

40 cases of hypertension were selected according to selection criteria.

SELECTION CRITERIA

1. Patients with systolic BP 140 mgHg or more, Diastolic BP 90 mmHg or more or taking antihypertensive drugs.
2. Patients of both sexes aged 41-60 years with or without associated risk factors –
    - Diabetes
    - Dyslipidemia

- Obesity and overweight
- Smoking
- Alcohol

EXCLUSION CRITERIA

Known reason for abnormal renal size

- Last cyst or multiple cyst
- Unilateral kidney
- End stage renal disease
- Hydronephrosis
- Renal tumors

All the cases of hypertension were thoroughly interrogate and clinically examined.

METHODS

History and clinical examination

All selected cases were subjected to detailed history recording and physical examination and data collected was noted serially in a predesigned proforma.

Blood pressure was recorded according to guidelines.

Patients Height (im mt.) and weight (in Kg.) were recorded.

Body mass index (BMI) of all cases was calculated by :

BMI = weight (in Kg) / Height^2 (in mt)

Classification	BMI (Kg/m^2)
Under weight	< 18.5
Normal range	18.5 – 24.9
Over weight	
Pre obese	25 – 29.9
Obese Class I	30 – 34.9
Obese Class II	35 – 39.9
Obese Class III	> 40

Body surface area (BSA) in $meter^2$ of all cases was calculated from standard table.

Investigations

Routine

1. Blood : Hb%
   TLC
   DLC
   ESR
   GBP
2. Urine : Routine
   Microscopic examination
   24 hr urinary protein
3. ECG

4. X-ray chest

5. Fundus examination by Ophthalmoscope

Specific

1. Blood Sugar — Fasting / Post Prandial

2. Serum creatinine

3. Lipid Profile : Total cholesterol, LDL, HDL, Triglyceride

4. Serum electrolytes (Na^+, K^+)

5. Real ultrasonography – For Kidney size

Ultrasonographic examination

Ultrasonographic examination of all patients were carried out with REAL TIME ULTRA SOUND SCANNING UNIT using 3.0 MHz frequency transducer. Thermal printer was used for print outs.

The patient were instructed to report to the department of Radiology in the morning. Ultra sonography of kidneys, ureter and bladder was done to measure kidney size and to exclude the case which were having other pathology of kidney like stone, polycystic kidney disease etc.

Mean values of measurements form the right and the left kidneys were used for statistical analysis. The data are presented as mean ± S.D. Student's two – tailed t-test was used to compare control and study groups. P values < 0.05 were considered statistically significant.

Univariate regression analysis was done to analyze the effect of following risk factor variables on kidney size :

- Blood pressure
- Body mass index (Kg/m^2)
- Body Surface Area (m^2)
- Fasting Blood Sugar (mg/dl)
- Lipid Profile : Total cholesterol (mg/dl)
  LDL (mg/dl)
  HDL (mg/dl)
  Triglycerides (mg/dl)
- Smoking
- Alcohol

Observation

OBSERVATIONS

TABLE I

DISTRIBUTION OF CASES ACCORDING TO AGE AND SEX

Age groups (yrs)	Male		Female		Total	
	No.	%	No.	%	No.	%
41-45	2	5.0	1	2.5	3	7.5
46-50	6	15.0	4	10.0	10	25
51-55	13	32.5	6	15.0	19	47.5
56-60	5	12.5	3	7.5	8	20
Mean±SD 52.08±5.5	26	65	14	35	40	100

Above mentioned table shows mean age of study group 52.08 ± 5.5 and total no. of male are 26. total no. of female are 14.

Percentage of male and female cases are 65% and 35%.

Maximum no. of cases belong to age group 51-55 years.

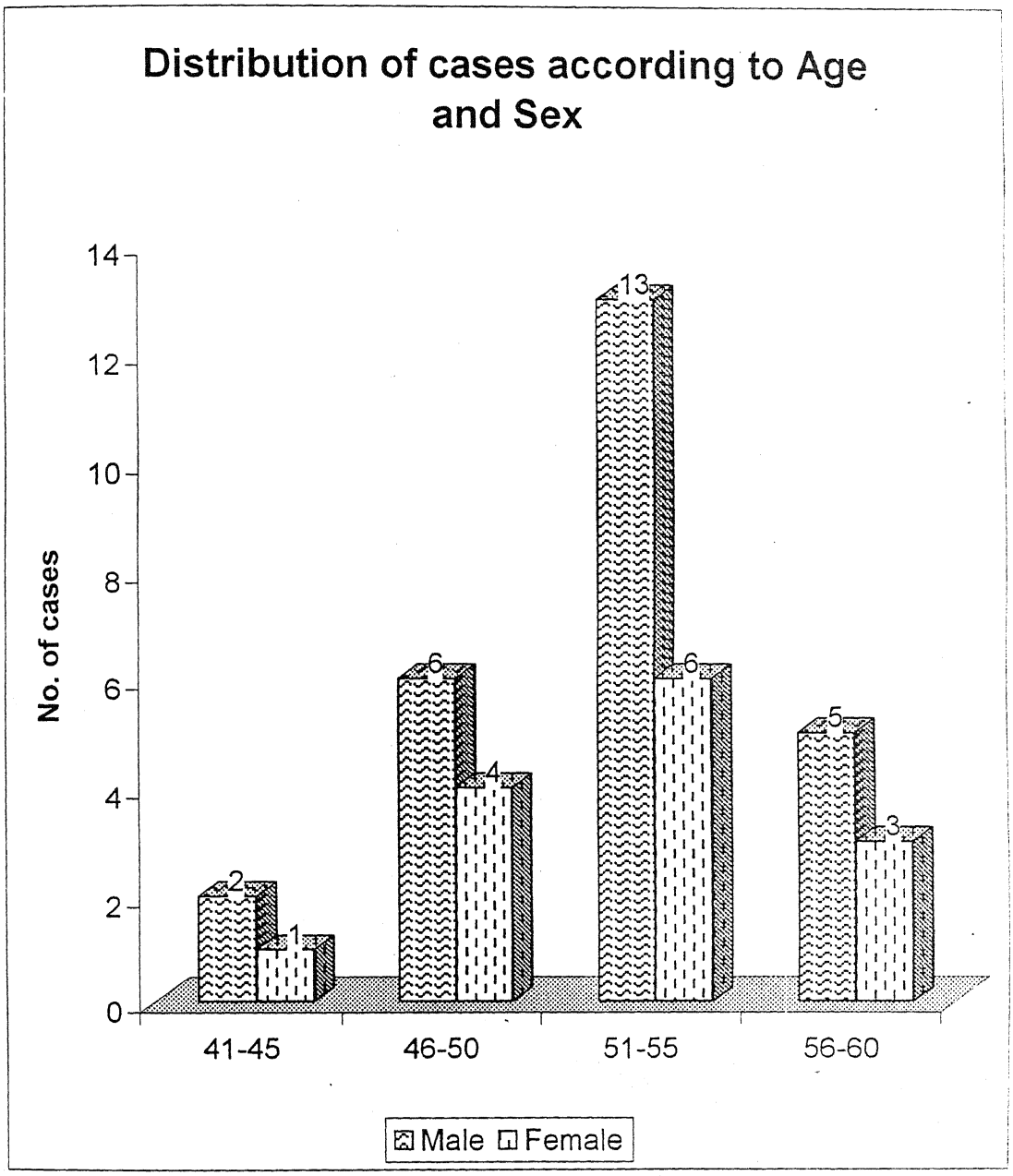

Distribution of cases according to Age and Sex
No. of cases
14
12
10
8
6
4
2
0
2
1
6
4
13
6
5
3
41-45
46-50
51-55
56-60
Male
Female

TABLE – II

DISTRIBUTION OF CASES ACCORDING TO RISK FACTORS IN MALE STUDY GROUP

S. No.	Risk factor	No. of cases	Percentage
1	Diabetes	14	53%
2	Dylipidemia ↑ Total cholesterol (>200mg/dl) ↑ LDL (>100mg/dl) ↑ Triglyceride (>150 mg/dl)	 13 5 11	 50 19.2 42.3
3	Overweight (BMI > 25.0)	18	69
4	Smoking	17	65.4
5	Alcohol	10	38.5

Total no. of cases in male study group = 26

- Above mentioned table shows 69% of cases in male study group were overweight (BMI>25.0), 65.4% were smokers and 53% of cases were diabetic.
- 50% of cases had increased total cholesterol (>200 mg/dl) and 42.3% had hypertriglyceridemia (triglyceride > 150 mg/dl).
- 38.5% cases were alcoholic.

Distribution of cases according to risk factors in male study goup

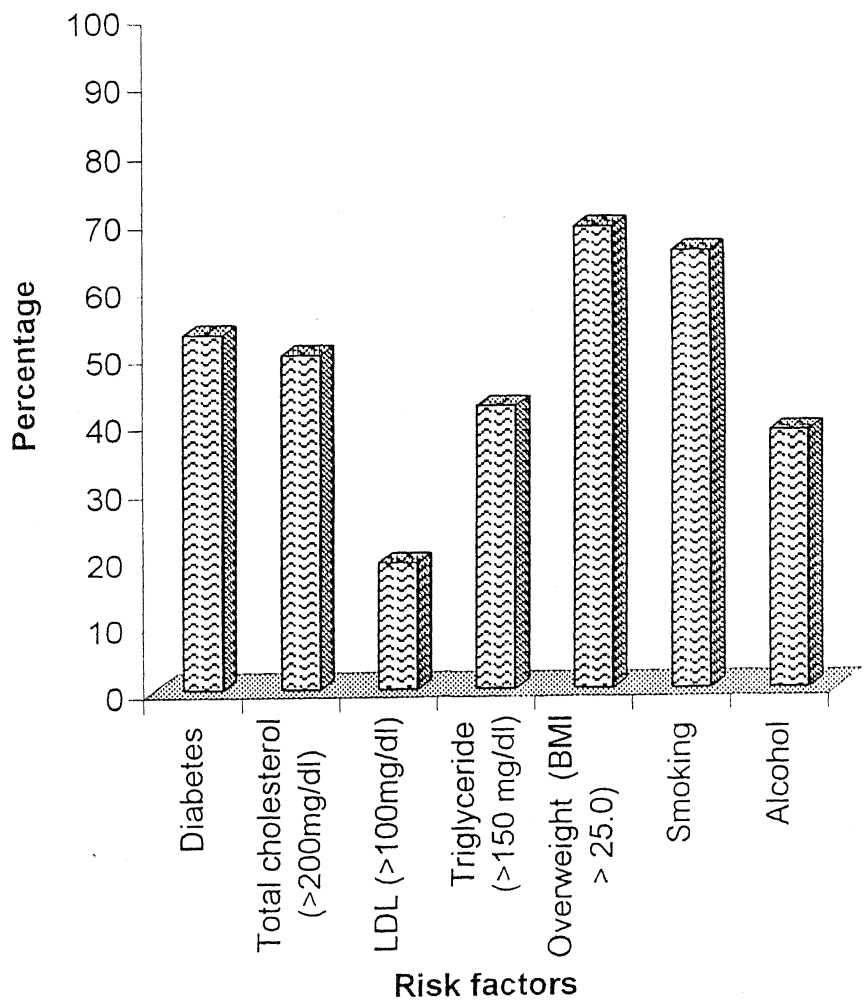


TABLE – III

DISTRIBUTION OF CASES ACCORDING TO RISK FACTORS IN FEMALE STUDY GROUP

S. No.	Risk factor	No. of cases	Percentage
1	Diabetes	6	42.9
2	Dylipidemia		
	↑ Total cholesterol (>200mg/dl)	8	57.14
	↑ LDL (>100mg/dl)	3	21.42
	↑ Triglyceride (>150 mg/dl)	6	42.9
3	Overweight (BMI > 25.0)	9	64
4	Smoking	-	-
5	Alcohol	-	-

Total no. of cases in female study group = 14

- Above mentioned table shows 64% of cases in female study group were overweight (BMI>25.0), 42.9% were diabetic.
- 57.1% cases had increased total cholesterol (>200 mg/dl) and 42.9% had hypertriglyceridemia.

Distribution of cases according to risk factors in female study goup

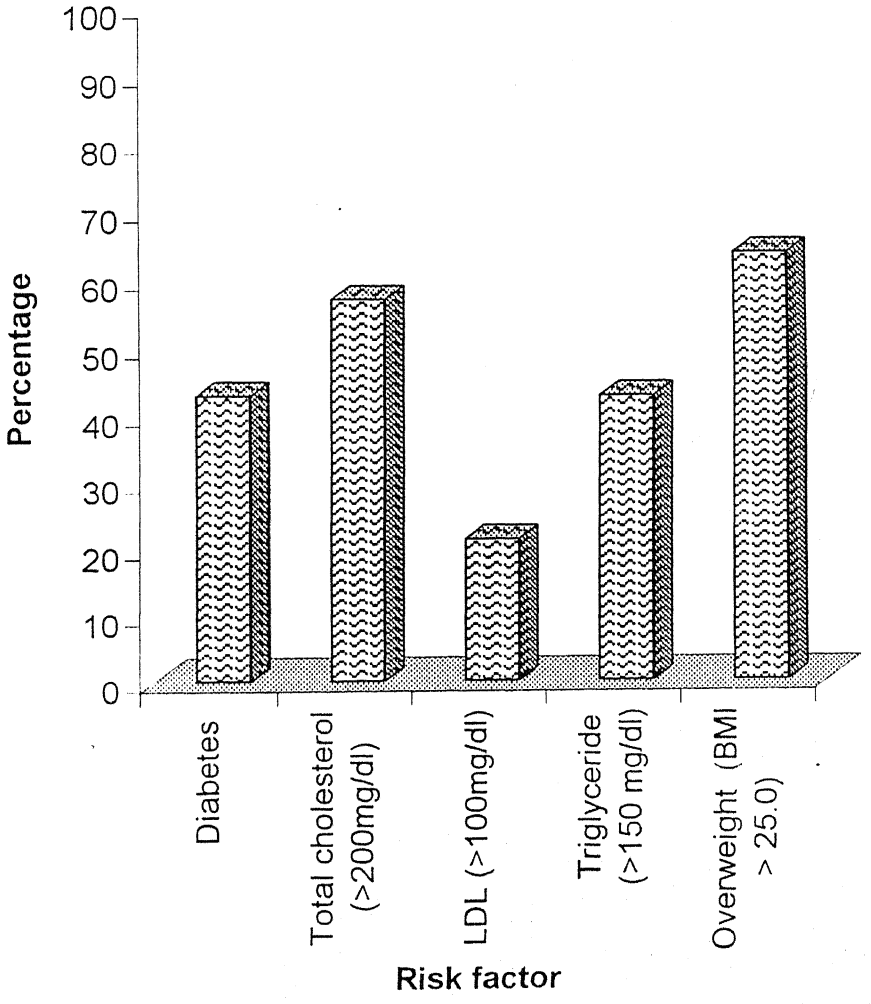


TABLE – IV

COMPARISON OF KIDNEY SIZE IN MALE CONTROLS AND STUDY GROUP

Group	No. of person	Mean Kidney (mm) size±S.D.	'p'
Control	10	109.85±1.65	< 0.05
Study group	26	111.44±1.77	

Above mentioned table shows the mean kidney size in control group in 109.85±1.65 and in study group 111.44 (mm) ±1.77.

Difference in mean kidney size in control and study group is statistically significant ($p < 0.05$).

Comparison of Kidney size in Male control & Study group

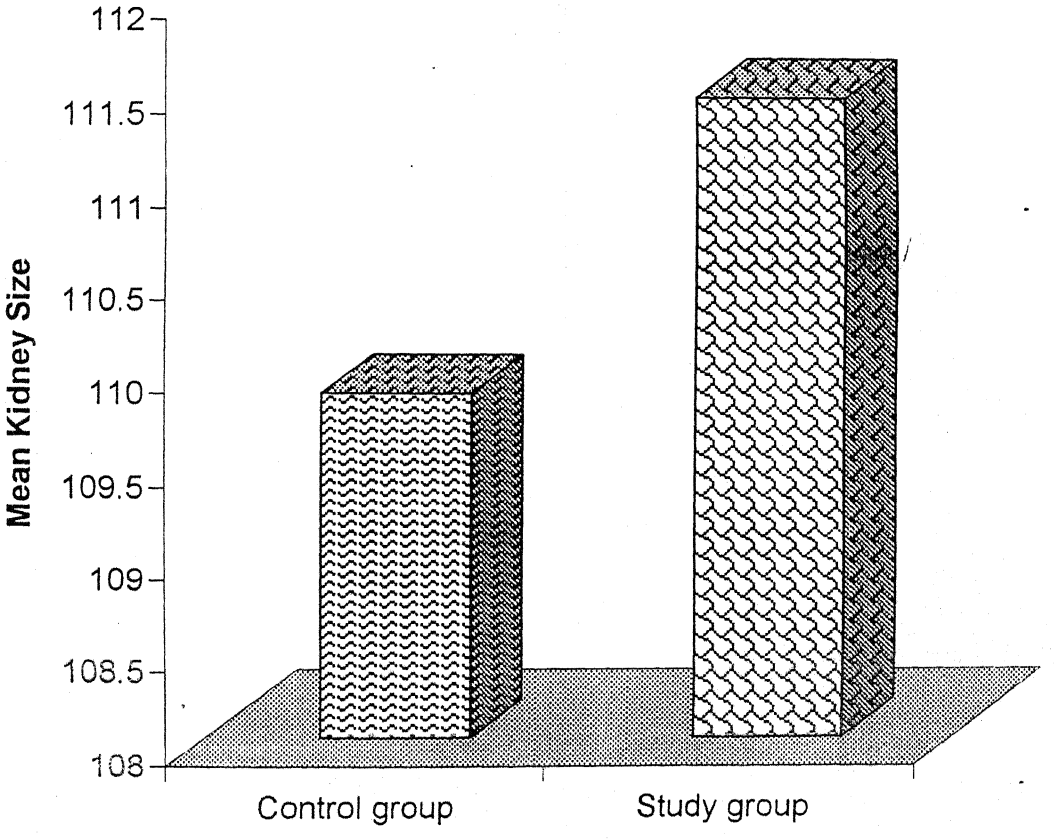


TABLE – V

COMPARISON OF KIDNEY SIZE IN FEMALE CONTROLS AND STUDY GROUP

Group	No. of person	Mean Kidney (mm) size±S.D.	'p'
Control	5	105.10±3.86	< 0.08
Study group	14	107.03±.3.52	

Above mentioned table shows the mean kidney size in control group in 105.1±3.85 and in study group 107.03 (mm) ±3.52.

Difference in mean kidney size in control and study group is borderline significant ($p < 0.08$).

Comparison of Kidney size in Female control & Study group

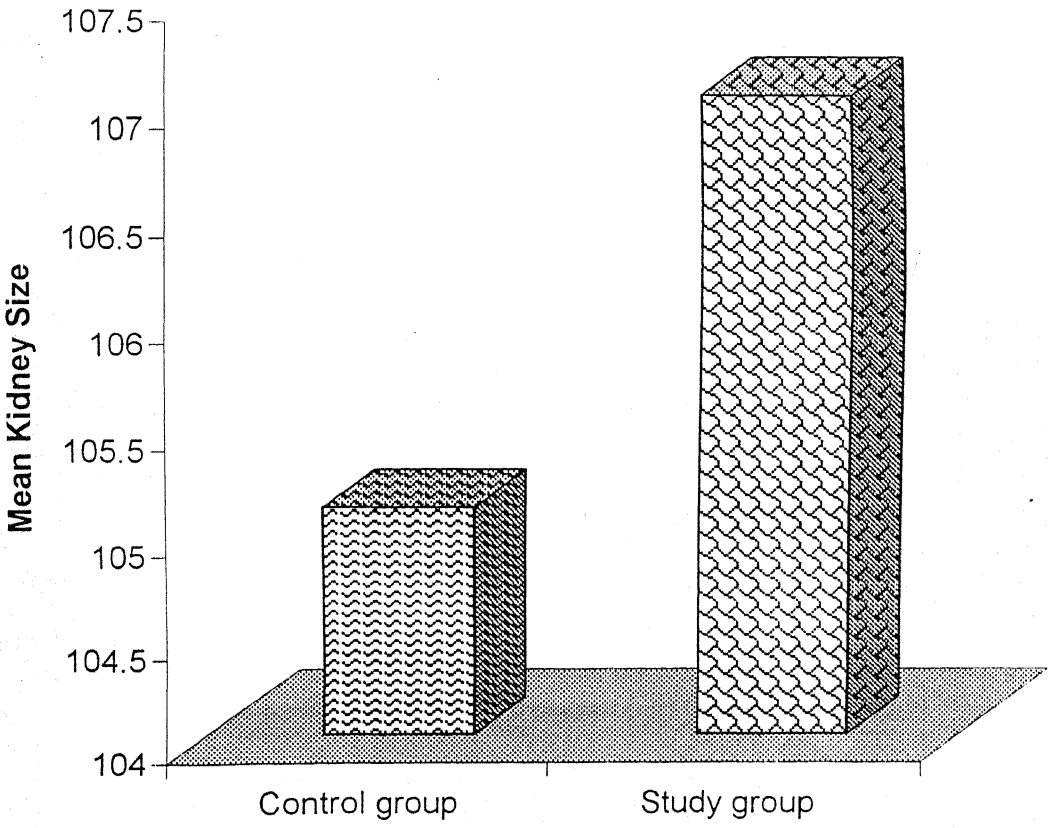


TABLE – VI

DISTRIBUTION OF CLINICAL DATA IN MALE CONTROL AND STUDY GROUP

Variables	Mean ± S.D.		'p'
	Control (n=10)	Study group (Hypertensive) (n=26)	
Age (yrs)	4.93 ± 3.56	51.96±3.8	> 0.05
BSA (m^2)	2.21±0.98	2.24±0.05	> 0.05
BMI (Kg/m^2)	25.82±0.99	25.6±0.66	> 0.05
Systolic B.P. (mmHg)	125±3.43	183.38±8.48	<0.00001
Diastolic B.P. (mmHg)	80.80±1.93	96.69±4.48	< 0.001
Fasting Blood Sugar (mmHg)	93±5.35	115.53±11.06	< 0.001
Total cholesterol (mg/dl)	178.20±0.97	204.26±12.61	< 0.001
LDL (mg/dl)	99.90±13.91	113.34±14.59	< 0.05
HDL (mg/dl)	48.50±7.51	51.61±10.49	< 0.05
Triglyceride (mg/dl)	130.30±26.70	197.15±13.12	< 0.001
Kidney size (mm)	109.85±1.65	111.44±1.77	< 0.05

(n=total no. of persons) ($p<0.05$ statistically significant)

- Above mentioned tables shows comparison of male control and study group.
- Difference in statistically significant in systolic B.P. ($p<0.00001$), diastolic B.P. ($p < 0.001$), fasting blood sugar ($p < 0.001$), total cholesterol ($p < 0.001$), LDL ($p < 0.005$), HDL ($p < 0.05$), triglyceride ($p<0.001$) and mean kidney size ($p < 0.05$).

TABLE – VII

DISTRIBUTION OF CLINICAL DATA IN FEMALE CONTROL AND STUDY GROUP

Variables	Mean ± S.D.		'p'
	Control (n=5)	Study group (Hypertensive) (n=14)	
Age (yrs)	49.4±3.43	52.35±4.14	> 0.05
BSA (m^2)	1.82±0.03	1.82±0.023	> 0.05
BMI (Kg/m^2)	24.72±3.0	26.02±1.46	> 0.05
Systolic B.P. (mmHg)	122.40±5.17	183±7.26	< 0.001
Diastolic B.P. (mmHg)	80.40±2.60	95.71±2.46	< 0.001
Fasting Blood Sugar (mmHg)	93±4	114.85±12.71	< 0.05
Total cholesterol (mg/dl)	175.20±8.43	205.14±14.88	< 0.05
LDL (mg/dl)	100.80±8.37	109.78±19.16	< 0.05
HDL (mg/dl)	48.50±7.51	49.2±9.63	> 0.05
Triglyceride (mg/dl)	142±25.17	199.57±5.09	< 0.05
Kidney size (mm)	105±3.86	107.03±3.52	< 0.08

(n=total no. of persons) (p<0.05 statistically significant)

- Above mentioned tables shows comparison of female control and study group.
- Difference in statistically significant in systolic B.P. ($p<0.001$), diastolic B.P. ($p < 0.001$), fasting blood sugar ($p < 0.05$), total cholesterol ($p < 0.05$), triglyceride ($p<0.05$) and mean kidney size ($p < 0.08$).

TABLE – VIII

REGRESSION ANALYSIS OF KIDNEY SIZE AND RISK FACTOR VARIABLES IN MALE STUDY GROUP

S. No.	Variable	'r'	'p'
1	Systolic BP	0.4150	0.03502
2	Diastolic BP	0.1409	0.49242
3	Body surface area (BSA)	0.7309	0.00002
4	Body mass index (BMI)	0.6974	0.00008
5	Blood sugar(fasting)	0.5144	0.00718 .
6	Total cholesterol	0.722	0.72590
7	LDL	-0.535	0.79514
8	HDL	0.1169	0.56947
9	Triglyceride	0.855	0.67803
10	Smoking	0.5305	0.02845
11	Alcohol	0.7488	0.01364

($p < 0.05$ statistically significant)

- Above mentioned table shows effect of risk factor variables on kidney size in male study group.
- Significant effect on kidney size is of BSA (p=0.00002) BMI (p=0.00008) and fasting blood sugar (p=0.00718).
- Statistically significant effect on kidney size was also observed in smokers (p=0.028) and alcoholics (p=0.013).

TABLE – IX

REGRESSION ANALYSIS OF KIDNEY SIZE AND RISK FACTOR VARIABLES IN FEMALE STUDY GROUP

S. No.	Variable	'r'	'P'
1	Systolic BP	-0.225	0.93910
2	Diastolic BP	-0.2201	0.44957
3	Body surface area (BSA)	0.8973	0.00001
4	Body mass index (BMI)	0.8755	0.0004
5	Blood sugar(fasting)	0.7419	0.00238
6	Total cholesterol	0.4417	0.11385
7	LDL	0.4760	0.8536
8	HDL	-0.2866	0.32045
9	Triglyceride	0.1697	0.56181

($p < 0.05$ statistically significant)

- Above mentioned table shows effect of risk factor variables on kidney size in female study group.
- Significant effect on kidney size is of BSA ($p=0.00001$) BMI ($p=0.0004$) and fasting blood sugar ($p=0.0023$).

Discussion

DISCUSSION

This study was a hospital based study and conducted at MLB Medical College, Jhansi. The aim of the study was to investigate whether the kidney size of hypertensive subjects differs form that of control subjects and whether the difference is due to hypertension per se or risk factors associated with hypertension.

Forty hypertensive patients, aged 41-60 years both male and female were selected from OPD, Nephrology clinic, Cardiology Clinic, hypertension clinic and inpatient wards. Controls were selected from same age group which were free from hypertension, renal diseases and diabetes.

In our study of middle aged adults mean age was 52.08 years with standard deviation ±5.5.

Mean age of male study group was 51.96 years with standard deviation of ±3.8 and of female study group was 52.96 years with standard deviation of ±4.14. Paivansalo MJ and Merikanto J (2001) reported mean age of male study group 50.6±5.9 and of female study group 51.6 ±5.6 years. In their study of renal size in hypertension.

In this study there was male preponderance. In study group 65% patients were male and 35% were female.

RISK FACTORS

In our study 69% male were overweight (BMI>25.0) while female in over weight category were 64%.

In male study group mean BMI was 25.90±0.66 while of control group mean BMI was 25.1±0.99. The difference was not significantly significant (p>0.005). In female, study group mean BMI was 26.02 ±1.46 while of control group mean BMI was 24.72 ± 3.0. The difference was statistically significant (p<0.05). Paivonsalo NIl, Merikanto J reported mean BMI 29.4 ± 4.4 in male study group and 28.7±5.3 in female study group in their study of renal size in hypertension.

In our study 53% male subjects were diabetic with mean fasting blood glucose 123.9±4.3 mg/dl while 42.9% female subjects were diabetic with mean fasting blood glucose 129 + 6.59 mg/dl. All male and female patients were an oral hypoglycemic agents.

Mean fasting blood glucose level of male hypertensive subjects was 115.53±11.06 and that of control group was 93 ± 5.35. The difference was statistically significant (p<0.05). Mean fasting blood glucose level of female hypertensive subjects was 114.85 ±12.71 and of control group was 93 ±4.0. The difference was statistically significant (p< 0.005).

In this study increased total cholesterol (>200mg/dl) was persent in 50% male study group. Mean total cholesterol in study group was 204.26±1.26, while in control group was 178.20±14.91. The difference was statistically significant ($p<0.05$). Increased total cholesterol was present in 57.14% female study group. Mean total cholesterol in female study group was 205.14±14.88 while in control group was 175.20±8.43. The difference was statistically significant ($p<0.05$).

Increased LDL (>130 mg/dl) was present in 19.2% male study group in our study. Mean LDL was 113.34±14.59 in male study group while in control group mean LDL was 99.90±13.91. The difference was statistically significant (<0.05). In female study group increased LDL was present in 21.42%. Mean LDL in female study group was 109.78±19.66 while in control group was 103.80±8.37. The difference was not statistically significant ($p>0.05$).

Increased triglyceride (>200 mg/dl) was present in 42.3% male study group in our study. Mean triglyceride in male study group was 197.15±13.12 while in control group was 150.30±26.70. The difference was statistically significant ($p<0.05$).

In female study group increased triglyceride level was present in 42.9%. Mean triglyceride level in female study group was 199.57±5.09

and in control group was 142±25.17. The difference was statistically significant ($p<0.05$).

In our study smoking and alcohol consumption was present only in male group. In male study group 65.4% were smoker and mean cigarette smoker per day were 15.4±7.3.

38.5% male patients were alcoholic and mean alcohol intake (ml/day) was 148±79.6.

KIDNEY SIZE :

	Observation	Author	Year
1	Men have larger kidney size than women	Karn Emamian	1962 1993
2	Size of right kidney is slightly smaller than left	Emamian	1993
3	No significant difference between size of Rt. and Lt. kidney	Buchholz NP & Abbas F	2000
4	Hypertensive subjects have larger kidney size than non hypertensive	Paivansalo MJ & Marikanto J	1998
5	No correlation between blood pressure and kidney size	Acran MM Oaivansalo KJ & Merikanto J Lane PH & Belsha CW	1989 1998 1998
6	Strong correlation between BSA and kidney size	Emamina Paivansalo MJ & Merikanto J	1993 1998
7	Strong correlation between BMI and kidney size	Emamian Painvansalo MJ & Merikanto J	1993 1998 2002

8	Diabetic patients (both IDDM & NIDDM) have larger kidney size than non diabetics	Avram MM & Hillary Hurtado Han Wirta O	1989 1980 1996
9	Smokers have larger kidney size than nonsmokers	Paivansalo MJ & Merikanto J	1998
10	Alcoholics have larger kidney size than nonalcoholics	Van Theil Paivansalo MJ & Merikanto J	1979 1998

In our study there was no significant difference between the sizes of right and the left kidney. Same results were observed by Buchholz NP & Abbas F, (2000). Emanian et al (19930 found the right kidney to be slightly smaller than the left in their study of healthy adult participants.

In our study men have larger kidneys than women in both control and study groups. Similar results were reported by Emamian et al (1993) AND Karn (1962) in their studies.

In our study hypertensive subjects had larger kidneys than control. Mean kidney size in male study group was 111.4±1.77 mm and in control group was 109.85±165 mm. The difference was statistically significant ($p<0.05$). Mean kidney size in female study group was 107.03±3.52mm and in control group was 105.10±3.86 mm. The difference was borderline significant (<0.08). The results are similar to the study conducted by Paivansalo MJ & Merikanto J (1998). They

reported mean kidney size in male hypetensive group 113.8±7.9 and in control group 111.6±7.3. Difference was statistically significant (p=0.002). In their study mean kidney size in female study group was 105.8±7.9 and in control group was 104.6±6.5, difference was borderline significant (p<0.08).

EFFECT OF VARIOUS RISK FACTORS ON KIDNEY SIZE :

In our study we included blood pressure, body surface area, body mass index, diabetes, dylipidemia, smoking and alcohol as risk factor variable.

By use of regression analysis we studied their effect on kidney size and found that body surface area, body mass index, diabetes smoking and alcohol significant effect on kidney size.

Effect of hypertension : In our study we observed that there was no effect of blood pressure on kidney size. Avram (1989) observed that there was no correlation between kidney size and blood pressure. Painvansalo MJ and Merikanto J (1998) reported the same observation in hypertensive adults. Lane PH & Belsha CW (1998) studied pediatric patients with essential hypertension and reported that there is no convincing correlation between kidney size and blood pressure.

EFFECT OF BODY SURFACE AREA :

Emamian et al (1993) Paivarisola MJ & Merikanto (1998), Buchholz NP & Abbas F (2000) observed strong correlation of body surface area with kidney size. Paivansalo MJ and Merikanto J (1998) studied significant effect of body surface area on kidney size. In our study we found significant effect of body surface area on kidney size (p=0.0002 for male group and 0.0001 for female group).

EFFECT OF BODY MASS INDEX :

Emamian et al (1993) Paivarisala MJ & Merikanto J (1998), Buchholz NP & Abbas F (2000) observed significant effect of body mass index on kidney size. In our study we also found significant effect of body mass index on kidney size (p=0.00008 for male study group and 0.0004 for female study group). Subjects with increased BMI had larger kidney size.

EFFECT OF DIABETES :

Avram MM & Hillary Hurtado, Han, Wirta et al (1996) have demonstrated that patients with NIDDM have large kidney size than non diabetic subjects. In our study 53% male subjects were diabetic (NIDDM). All patients were receiving oral hypoglycemic agents. Mean kidney size in diabetic male subjects was 112.6±1.7 mm while in non diabetic subjects was 110.1±0.83 mm. The difference was statistically

(NIDDM) and all were receiving oral hypoglycemic agents. Mean kidney size of diabetic female subjects was 109.42±1.88 mm and of non diabetic was 105.25±3.62 mm. The difference was statistically significant ($p<0.05$). By regression analysis we observed that fasting blood glucose level had significant effect on kidney size ($p=0.007$). larger kidney size observed in subjects with higher fasting blood glucose level.

EFFECT OF DYSLIPIDEMIA :

We studied effect of increased total cholesterol, increased LDL, HDL level and increased triglycerides on kidney size and observed no significant effect of above risk factor variables on kidney size.

EFFECT OF SMOKING AND ALCOHOL :

Pavansolo MJ, Merikanto J (1998) observed larger kidney size in smokers. In our study 65.4% male subjects were smoker with mean cigarettes smoked per day of 15.4±7.27. Mean kidney size in smokers was 111.2±1.21 and in non smokers subjects was 108.7±1.35. The difference was statistically significant ($p<0.001$). Renal size increased with number of cigarettes smoked per day. On regression analysis we observed significant effect of smoking on kidney size ($p=0.02$).

Van Thiel et al observed renal hypertrophy after chronic alcohol ingestion in rats. Pavansolo MJ and Merikanto J (1998) observed larger

kidneys in alcohol users in middle aged adults. In our study 38.5% male subjects were alcohol users with mean alcohol intake (ml/day) 148±79.7. Mean kidney size in alcohol uses was 111.2±1.08 and in teatotalle subjects was 108.70±1.35. The difference was statistically significant ($p<0.05$). On regression analysis we observed significant effect of alcohol on kidney size ($p<0.13$).

Conclusion

CONCLUSION

Hypertension is one of the most important health problem in the world and it affects Heart, Kidneys, Eyes, Brain and almost all organs of the body, various risk factors are associated with hypertension these are mainly Diabetes, Obesity, Dyslipidemia, Smoking and Alcohol.

This study was done in middle aged adults to analysed whether kidney size is affected by hypertension per se or risk factors associated with hypertension. The renal measurements were performed by abdominal ultrasound. Genders were analysed separately. Following conclusions have been derived.

- Maximum number of cases belonged to 51-55 years and their mean age was 52.08±5.5 years.
- There was male preponderance 65% cases were male and 35% were female.

MALE STUDY GROUP :

- Hypertensive men had larger kidney size (111.44±1.77 mm) than control (109.85±1.65) and difference was statistically significant ($p<0.05$).
- Blood pressure did not have significant effect on renal size ($p>0.05$).

- Body surface area had significant effect on renal size (p>0.0002) larger kidney size observed in subjects having more body surface area.
- Body mass index had significant effect on kidney size (p=0.00008). 69% male subjects were overweight (BMI > 250) in our study. Larger kidney size was observed in subjects having increased BMI.
- Diabetic subjects had larger kidney size (112.6+1.7 mm) than non diabetic subjects (110.1 + 0.83 mm). The difference was statistically significant. Fasting blood glucose had significant effect on kidney size (p=0.007). Larger kidney size was observed in subjects with higher fasting blood glucose level.
- Lipid profile abnormalities were observed in hypertensive male subjects. Increased total cholesterol (>200mg/dl) was present in 50% cases, increased LDL (>100 mg/dl) was presenting 19.2% cases and increased Triglyceride level (>150 mg/dl) was observed in 42.3% cases. No significant effect of lipid abnormalities on kidney size was observed.
- Smokers had larger kidney size (111.2±1.21 mm) than non smokers(108.7±1.35 mm). The difference was statistically significant (p<0.05). kidney size increased with number of

cigarettes smoked per day. On regression analysis smoking had significant effect on kidney size (p=0.02).

- Significant effect of alcohol was observed on kidney size (p=0.01). Alcoholic subject had larger kidney size.

FEMALE STUDY GROUP :

- Hypertensive women had larger kidney size (10.7.03±3.52 mm) than control (105.10±3.86). The difference was borderline significant (p<0.08).
- No effect of blood pressure on kidney size observed in female study group.
- As in female study group, female subjects with more body surface area had larger kidney size and the effect was highly significant (p=0.0001).
- 64% female were over weight (BMI > 25.0). BMI had significant effect on kidney size (p=0.004). Larger kidney size was observed in female subjects with increased BMI.
- In female study group diabetic subjects had larger kidney size (109.42±1.88 mm) than Non diabetic subjects (105.25±3.62 mm). The difference was statistically significant (p<0.05). Fasting blood glucose label had significant effect on kidney size

(p=0.002). Larger kidney size observed in subjects with higher fasting blood glucose level.

- Lipid profile abnormalities were also observed in female hypertensive subjects. Increased total cholesterol was observed in 57.14%, increased LDL was present in 21.42% and increased triglyceride was present 64% subjects. No significant effect of lipid profile abnormalities observed on kidney size.

We conclude that significant factors affecting kidney size were body surface area, body mass index, diabetes, smoking and alcohol. The hypertensive subjects had larger kidney size than the controls mainly because more frequent associated overweight, abnormal blood glucose test values, smoking and alcohol intake.

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Appendix

APPENDIX - I

WORKING PROFORMA

EFFECT OF HYPERTENSION, AND ASSOCIATED RISK FACTORS ON KIDNEY SIZE IN MIDDLE AGED ADULT

Case No.: ..... Date: .....

1. Patient Name
2. Age/sex
3. OPD/Ward/Bed
4. Occupation
5. Address
6. Diagnosis

- Chief complaints and Brief History
- Past History
- Treatment History
- Personal History

EXAMINATION

A. General

- Pulse
- Blood pressure
- Respiration
- Temperature

- Pallor/cyanosis/ Icterus/ Pedal Oedema/ Facial puffiness/ JVP/ Clubbing

B. Body Surface Area (BSA)

C. Body Mass Index (BMI, Kg/m^2)

D. Systemic

- Cardiovascular
- Respiratory
- Nervous system
- Abdomen
- Any other

Investigations

- Hb%
- TLC, DLC
- ESR
- GBP
- Blood sugar

Fasting Post prandial

- Serum creatinine
- Lipid profile
- Serum Electrolytes (Na^+, K^+)
- Urine Examination

Routine Microscopic 24 hr. urinary protein

- Fundus examination by ophthalmoscope
- ECG

Ultra sound K.U.B. region

- Renal size

	Left	Right
Length (mm)		
Width (mm)		

- Corticomedullary differentiation
- Echogenicity

- Other relevant investigation as per case

APPENDIX - II
DATA STUDY GROUP

S.No.	Name	Age (yrs)	Sex	Smoking Cigarettes Per day	Alcohol (mg/day)	Diabetes	Systolic B.P. (mmHg)	Diastolic B.P. (mmHg)	BSA (m^2)	BMI (Kg/m^2)	Fasting Blood Glucose (mg/dl)	P.P. Blood Glucose (mg/dl)	Serum Creatinine (mg/dl)	Total cholesterol (mg/dl)	LDL (mg/dl)	HDL (mg/dl)	Triglyceride (mg/dl)	Kidney size (mm)		
																		Rt.	Lt.	Mean
1.	SL	52	M	16	250	-	184	100	2.28	25.9	106	144	0.9	220	115	64	204	111	113	112
2.	Rani	51	F	-	-	-	176	96	1.84	25.8	128	182	1.1	208	110	58	200	108	109	108.5
3.	MM	48	M	-	-	-	180	96	2.30	26.2	124	182	0.8	196	107	48	218	112	115	113.5
4.	VD	54	F	-	-	-	186	98	1.80	25.00	116	146	1.0	190	91	50	192	104	106	105.0
5.	VL	50	M	9	130	-	170	90	2.16	24.40	92	144	0.8	216	113	61	212	109	110	109.5
6.	SD	55	F	-	-	-	178	94	1.82	24.40	104	144	0.9	226	117	48	207	106	108	107
7.	RKG	58	M	8	60	-	190	100	2.26	24.90	104	136	0.8	188	89	60	195	109	110	109.5
8.	JR	54	M	-	-	-	196	108	2.20	25.80	118	202	1.0	218	114	64	202	110	110	110
9.	RD	55	F	-	-	-	188	98	1.78	23.20	92	146	1.1	196	90	46	200	97	98	97.5
10.	HD	45	F	-	-	-	184	96	1.82	26.40	128	208	0.7	190	91	60	196	108	108	108
11.	RL	51	M	28	-	-	196	102	2.30	28.20	128	168	0.7	222	118	66	206	113	116	114.5
12.	Sharda	54	F	-	-	-	174	94	1.80	24.80	108	130	1.1	188	89	40	195	102	104	103
13.	AS	55	M	17	-	-	172	92	2.26	26.20	120	164	0.9	194	88	66	202	111	112	111.5
14.	CP	52	M	15	150	-	184	96	2.22	26.00	116	140	0.9	219	115	62	232	109	110	109.5
15.	PNS	46	M	-	-	-	192	92	2.24	29.10	126	174	0.8	196	89	68	197	115	117	116.0
16.	SK	58	F	-	-	-	180	96	1.82	27.00	96	124	1.1	216	133	42	206	106	107	106.5
17.	LR	45	M	-	-	-	168	92	2.24	24.70	90	132	1.0	226	117	68	207	110	111	110.5
18.	RD	52	M	20	-	-	170	90	2.26	26.80	126	148	1.0	190	89	62	195	112	112	112
19.	JPS	58	M	18	250	-	188	98	2.24	25.60	114	142	0.9	208	111	57	199	111	111	111
20.	Jagmati	58	F	-	-	-	190	98	1.84	24.80	130	172	1.2	210	106	58	201	108	109	108.5
21.	Omkali	57	F	-	-	-	170	90	1.86	28.80	118	168	1.0	196	90	56	200	110	110	110
22.	VB	48	F	-	-	-	188	96	1.86	28.00	138	162	0.9	226	128.6	54	197	111	113	112
23.	RP	58	M	26	-	-	196	100	2.28	27.20	132	174	1.1	197	108.6	47	208	113	113	113
24.	SKS	50	M	-	180	-	178	98	2.24	24.60	98	132	1.0	189	108.8	41	196	110	111	110.5
25.	RK	52	M	20	-	-	186	98	2.26	26.00	116	206	0.8	212	141.6	37	167	112	112	112
26.	GS	47	M	24	-	-	192	100	2.30	27.00	128	164	0.9	208	124.2	46	189	113	113	113
27.	VA	48	M	-	50	-	180	94	2.16	23.90	102	146	0.8	198	109.8	51	186	109	110	109.5
28.	VK	53	M	-	-	-	182	92	2.14	24.80	108	147	0.9	180	97.2	50	160	109	109	109
29.	RR	43	M	8	-	-	176	96	2.22	24.70	122	176	1.1	209	121.4	41	198	111	111	111
30.	MA	57	M	-	150	-	184	92	2.16	25.80	116	138	1.0	228	135	44	202	110	111	110.5
31.	AR	50	F	-	-	-	186	96	1.84	26.60	132	170	1.1	232	151.4	39	208	109	110	109.5
32.	Sharla	48	F	-	-	-	194	94	1.80	25.80	108	138	0.9	200	113.6	47	197	106	106	106
33.	Islama	52	F	-	-	-	184	100	1.84	26.80	106	136	0.8	191	104.2	49	189	108	109	108.5
34.	RA	55	M	6	-	-	192	104	2.28	26.00	124	164	0.9	226	138	38	201	112	112	112
35.	BL	55	M	18	180	-	180	96	2.24	26.40	108	136	1.1	196	118.5	39	191	110	111	110.5
36.	KPS	51	M	5	-	-	176	92	2.30	26.00	126	148	1.0	189	113.2	42	169	112	113	112.5
37.	SRM	45	M	-	80	-	182	96	2.18	25.00	96	130	1.1	200	117	44	200	110	110	110
38.	CM	57	M	18	-	-	192	100	2.32	26.00	124	186	1.0	214	126.2	47	204	114	114	114
39.	LPS	52	M	6	-	-	180	98	2.20	26.20	120	186	0.7	224	142	39	212	111	111	111
40.	Saroj	48	F	-	-	-	188	94	1.82	25.80	118	158	1.1	224	133	42	201	108	109	108.5

APPENDIX - III
DATA CONTROL GROUP

S.No.	Name	Age (yrs)	Sex	Smoking Cigarettes Per day	Alcohol (mg/day)	Diabetes	Systolic B.P. (mmHg)	Diastolic B.P. (mmHg)	BSA (m^2)	BMI (Kg/m^2)	Fasting Blood Glucose (mg/dl)	P.P. Blood Glucose (mg/dl)	Serum Creatinine (mg/dl)	Total cholesterol (mg/dl)	LDL (mg/dl)	HDL (mg/dl)	Triglyceride (mg/dl)	Kidney size (mm)		
																		Rt.	Lt.	Mean
1.	RK	48	M	15	-		124	84	2.26	25.80	92	130	0.8	188	89	60	146	111	112	111.5
2.	NP	52	M	8	200	-	132	82	2.12	24.30	94	141	0.9	184	94	40	124	109	110	109.5
3.	S	50	F	-	-	-	118	78	1.84	26.00	92	152	1.1	172	100	47	140	106	108	107
4.	P	48	F	-	-	-	128	80	1.86	25.4	98	138	0.8	166	90	44	142	108	109	108.5
5.	RP	45	M	26	250	-	134	80	2.30	26.40	100	142	0.9	152	91	38	118	112	113	112.5
6.	LK	50	M	-	-	-	128	80	2.20	25.80	96	152	1.0	164	84	49	150	108	109	108.5
7.	R	46	F	-	-	-	120	78	1.82	24.60	96	160	0.9	182	102	52	138	106	106	106
8.	JS	45	M	6	150	-	130	84	2.24	25.20	102	148	0.8	192	98	48	146	109	109	109
9.	SK	55	M	12	-	-	122	78	2.28	25.50	91	130	0.9	200	86	51	148	110	111	110.5
10.	PN	46	F	-	-	-	116	80	1.78	24.20	88	132	0.8	170	92	50	112	105	106	105.5
11.	SK	48	M	-	-	-	136	86	2.26	26.10	84	142	0.9	172	99	46	141	110	110	110
12.	PN	44	M	-	50	-	128	82	1.98	22.40	89	136	0.8	178	97	42	138	106	107	106.5
13.	RN	48	M	25	80	-	132	78	2.26	25.10	93	143	0.9	164	87	51	131	110	110	110
14.	PK	49	M	16	180	-	126	82	2.28	24.60	89	151	1.0	188	89	60	136	110	111	110.5
15.	RN	54	F	-	-	-	136	88	1.80	23.50	91	141	0.9	186	96	48	138	98	99	98.5